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DOI:10.1038/ijo.2011.5

Int J Obes (Lond). 2011 Nov;35(11):1442-9. doi: 10.1038/ijo.2011.5. Epub 2011 Feb 1

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Running title: Contributors to obesity and hyperglycemia epidemics

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Grant: This study was supported by a grant from: Regione Piemonte, 2008.

Word count: abstract 250, text 2777, 4 tables, 2 figures

Abstract

Objective: Relatively unexplored contributors to the obesity and diabetes epidemics may include: sleep restriction, increased house temperature (HT), television watching (TW), consumption of restaurant meals (RM), use of air conditioning (AC) and use of antidepressant/antipsychotic drugs (AD). *Design and Subjects:* In a population based-cohort (*n*=1597), we investigated the possible association among these conditions and obesity or hyperglycemia incidence at six-year-follow-up. Subjects with obesity (*n*=315) or hyperglycemia (*n*=618) at baseline were excluded: respectively 1282 and 979 individuals were therefore analyzed. *Results:* At follow-up, 103/1282 became obese; these subjects showed significantly higher BMI, waist circumference, saturated fat intake, RM frequency, TW hours, HT, AC and AD use and lower fiber intake, metabolic equivalent of activity in hours/week (METS) and sleep hours at baseline. In a multiple logistic regression model, METS (OR=0.94;95%CI 0.91-0.98), RM (OR=1.47 per meal/week;1.21-1.79), being in the third tertile of HT (OR=2.06; 1.02-4.16) and hours of sleep (OR=0.70 per hour; 0.57-0.86) were associated with incident obesity. Subjects who developed hyperglycemia (*n*=174/979; 17.8%) had higher saturated fat intake, RM frequency, TW hours, HT, AC and AD use at baseline and lower METS and fiber intake. In a multiple logistic regression model, fiber intake (OR=0.97 for each g/day; 0.95-0.99), RM (1.49 per meal/week; 1.26-1.75) and being in the third tertile of HT (OR=1.95; 1.17-3.26) were independently associated with incident hyperglycemia.

Conclusions: Lifestyle contributors to the obesity and hyperglycemia epidemics may be regular consumption of RM, sleep restriction and higher HT, suggesting potential adjunctive nonpharmacologic preventive strategies for the obesity and hyperglycemia epidemics.

Key words: obesity, hyperglycemia, restaurant meals, sleep restriction, home temperature

Introduction

Obesity and diabetes have reached epidemic proportions in the United States and Europe. Changes in dietary habits and reductions in physical activity are the two most common explanations for the increasing burden of these diseases. Recently, attention has been directed to other contributors, that may influence the balance between energy expenditure and intake [1-8]. Furthermore, dietary and exercise habits are not easily modifiable, and the search for risk factors that are more amenable to change might be potentially interesting and relatively unexplored in European cohorts.

The average amount of sleep per night has been reported to be declining slightly [9] and an increased incidence of obesity and hyperglycemia has been noted along with this decrease in sleep, particularly in younger cohorts [10]. This finding is probably due to the profound metabolic hormonal changes exerted by sleep debt and increased fatigue, leading to increased caloric intake and reduced energy expenditure, respectively [2-3, 10]. Other features of modern societies include psychosocial stress, indoor heating during cold seasons and air conditioning during warm seasons.

The use of antidepressants and atypical antipsychotics that are associated with weight gain has increased substantially in the last decade [1]. These conditions, together with increasing television watching and consuming foods that have been prepared outside the home, might promote a decline in physical activity and overeating [4, 6].

In a population based-cohort we investigated possible associations among sleep restriction, house temperature, hours of television watching, consumption of restaurant foods, use of air conditioning, use of antidepressant/antipsychotic drugs and the risk of obesity and hyperglycemia, after an average follow-up period of six-years.

Subjects and Methods

All 1,877 Caucasian patients aged 45-64 years of six family physicians were invited to participate in a metabolic screening between 2001 and 2003. These subjects were representative of the Local Health Units of the province of Asti (North-western Italy) as reported previously [11]. In total, 1658 patients (88.3%) agreed to participate by written informed consent, whereas 219 declined. Both participants and non-participants showed the same gender distribution, level of education, prevalence of known diabetes and subjects living in rural areas as the resident population of the corresponding age-group, in the same area [11]. Clinics were held in the morning after fasting overnight; for each patient, weight, height, waist circumference (measured by a plastic tape meter at the level of the umbilicus) and blood pressure were measured, and a fasting blood sample was drawn. Systolic and diastolic blood pressures were measured twice with a standard mercury sphygmomanometer with the patient in a sitting position after at least 10 min of rest. The reported values are the means of two measurements.

All patients answered a questionnaire at the health screening. The following data were collected for each subject: smoking habits, alcohol consumption, education level, health conditions, drugs used, sleep duration, mean house temperature during autumn/winter, mean daily number of hours of television watching, mean weekly number of meals consumed in restaurants (also considering fast-food restaurants and pizzerias) and regular use (>2 days/week) of air conditioning during the summer season. Sleep duration was defined as self-reported time in bed (calculated from bedtime to rise time) minus sleep latency.

All subjects completed the validated, semi-quantitative food-frequency questionnaire used in EPIC (European Prospective Investigation into Cancer and Nutrition) studies [12] and the Minnesota-Leisure-Time-Physical-Activity questionnaire [13].A dietician who was blinded to the study details checked all questionnaires for completeness, internal coherence and plausibility. Each nutrient was adjusted for total energy using the residual method [14]. The leisure physical activity level was calculated as the product of the duration and frequency of each activity (in hours/week), weighted by an estimate of the metabolic equivalent of the activity (MET), and summed for all activities performed.

From January to November 2008, patients were contacted for follow-up visits. Deaths occurred in 61/1658 (3.7%) subjects during the follow-up period. All the remaining 1597 patients had weight, waist circumference and blood pressure measurements taken, and a blood sample was drawn for the determination of fasting metabolic parameters.

All procedures were in accordance with the Declaration of Helsinki. The study was approved by the local Ethics Committee.

Laboratory methods have been described previously [11]. Diabetes and impaired fasting glucose (IFG) were defined in accordance with guidelines [15].

Statistical analyses

When analyzing the association between the incidence of obesity and baseline variables, subjects with obesity (BMI≥30 kg/m^2) at baseline (n=315/1597; 19.7%) were excluded. When analyzing the association between the incidence of hyperglycemia and baseline variables, subjects with hyperglycemia (fasting glucose ≥5.6 mmol/L) at baseline (n=618/1597; 38.7%) were excluded. Therefore, analyses were performed in 1282 and 979 subjects, respectively. Due to the low number of expected incident cases of type 2 diabetes, our study did not have sufficient statistical power to detect differences in baseline variables between patients with and without incident diabetes. Therefore, incident IFG and diabetes were combined into one category, termed incident hyperglycemia.

A Student's t test (normal distribution) or Mann-Whitney test (skewed distribution) and a χ^2-test were performed to assess raw differences in baseline continuous and categorical variables, respectively. A logistic regression analysis was performed to estimate adjusted odds ratios among baseline fiber intake, saturated fat intake, METS, degrees centigrade (°C) of house temperature, hours of sleep, hours of television watching, mean number of restaurant meals per week, antidepressant/antipsychotic drug use, air conditioning use and incident obesity and incident hyperglycemia, after controlling for sex, baseline BMI, education level and (in cases of incident hyperglycemia) baseline glucose values and alcohol intake. A multiple linear regression model was conducted to evaluate the association among these variables and continuous values of BMI and fasting glucose at follow-up.

Due to the suspect of a non-linear relationship between incremental house temperature and BMI and fasting glucose at follow-up, tertiles of house temperature were used as dummy variables, using the lowest tertile as a reference.

Results

Incident obesity

Baseline characteristics according to obesity development are reported in Table 1. At follow-up, 103/1282 (8.0%; 95%CI 6.5-9.5) subjects had become obese; those subjects showed significantly higher values of BMI, waist circumference, saturated fat intake, frequency of restaurant meals, hours of television watching, house temperature, use of air conditioning and antidepressant/antipsychotic drugs at baseline. They also had significantly lower fiber intake, METS and hours of sleep.

BMI values at follow-up showed an overall linear increase with increasing numbers of restaurant meals, hours of television and reduced hours of sleep, fiber intake and METS (Figure 1). The mean BMI at follow-up was highest in the small group of subjects consuming ≥4/restaurant meals/week: 1.72kg/m^2 (corresponding to a 3kg increase in weight and a 4cm increase in waist circumference).

In a multiple logistic regression model, after adjusting for sex, education level, baseline BMI and all the variables listed in Table 3, the following variables were independently associated with incident obesity: reduced level of exercise, increased number of restaurant meals (for each additional meal per week), being in the highest tertile of house temperature and reduced hours of sleep (Table 3).

When using BMI at follow-up as a continuous variable, both a reduced level of exercise (β= -0.02; 95%CI –0.03 to -0.01, $p<0.001$) and an increased number of restaurant meals (β=0.27; 95%CI 0.19 0.35, $p<0.001$ for each additional meal per week) remained significantly associated with BMI at follow-up, after carrying out a multiple linear regression model that was adjusted for all the variables listed in Table 3.

Incident hyperglycemia

Baseline characteristics according to hyperglycemia development are reported in Table 2. At follow-up, 174/979 (17.8%; 95%CI 15.4-20.2) subjects had developed hyperglycemia; those subjects were more frequently male and had significantly higher values of waist circumference, fasting glucose, saturated fat intake, alcohol intake, mean number of restaurant meals per week, hours of television watching, house temperature and use of air conditioning and antidepressant/antipsychotic drugs at baseline. They also showed significantly lower fiber intake and exercise levels.

Fasting glucose values at follow-up increased with increasing numbers of restaurant meals per week and hours of television watching as well as reduced fiber intake (Figure 2). The greatest increase in fasting glucose values during follow-up was 1.2 mmol/L in the group eating ≥4 restaurant meals/week (incident hyperglycemia=61%; 95%CI 43-79), whereas the lowest (-0.02 mmol/L) was in the group with the highest fiber intake (incident hyperglycemia=14.5%; 95%CI 10.6-18.4).

In a multiple logistic regression analysis, after adjusting for sex, education level, alcohol intake, baseline BMI and glucose and all the variable listed in Table 4, the following variables were independently associated with incident hyperglycemia: reduced fiber intake, increased number of restaurant meals (for each additional meal per week) and being in the highest tertile of house temperature (Table 4). When using glucose level at follow-up as a continuous variable, both reduced fiber intake ($\beta = -0.008$; 95%CI –0.012 to -0.004, $p=0.001$) and increased number of restaurant meals per week ($\beta=0.22$; 95%CI 0.18 0.26, $p<0.001$ for each additional meal per week) remained significantly associated with glucose values at follow-up after performing a multiple linear regression model that was adjusted for all the variables reported in Table 4.

A sensitivity analysis was performed in all patients including the 61 subjects who had died during the follow-up period, applying two extreme scenarios: assuming that either none of them or all of them had developed obesity or hyperglycemia at the follow-up. These results were consistent with those obtained when analyzing the living individuals only.

Discussion

Alternative independent contributors to the obesity and hyperglycemia epidemic, other than physical inactivity and increased energy intake, may be the regular consumption of restaurant meals, sleep restriction and higher home temperature. Other factors, such as television watching and the use of antidepressant/antipsychotic drugs and air conditioning, may have a lesser impact.

Sleep restriction

Sleep duration has declined from 8-9 hours per night to 7 hours or less per night in the last 50 years, largely as a consequence of voluntary sleep restriction (watching television, using the

Internet and getting more work done, etc.) [1]. Sleep debt is associated with decreased rates of glucose clearance, insulin response and glucose effectiveness, increased sympathetic nervous system activity and impaired glucose regulation by reduced lipolytic effects [16]. During sleep restriction, plasma leptin levels are decreased, whereas ghrelin, cortisol and orexin secretion is increased [1, 16]. Thus, the link between sleep debt and hormones implicated in feeding regulation explains the observed increase in appetite and food intake [17], particularly for energy-dense, high-carbohydrate foods [16]. This finding and the observed decrease in daytime physical activity after sleep loss [18] may contribute to the documented increased risk of obesity [2, 19]. However, these associations have been criticized because the epidemiological evidence is weak and the risk seems very small and develops over many years in very short sleepers (around 5 hours) [20-21]. The relationships between sleep duration and incident diabetes are contrasting, since large US epidemiological studies have found associations between both short and long sleep duration and diabetes [3, 22-23]. Associations have been found to be significant only in a subset of diabetic patients with severe symptoms, and not for short sleepers after adjusting for BMI [22], in men but not in women [24], and other studies failed to find any association [25]. On the other hand, a growing number of epidemiological studies and meta-analyses have provided evidence of an association between short-duration sleep and the risk of obesity, as reviewed [1, 16]. In accordance with the literature, in our cohort, sleep restriction was associated with obesity at follow-up, but not with incident hyperglycemia at follow-up.

Indoor temperature

The ability of brown adipose tissue (BAT) to burn rather than store calories depends on its mitochondrial uncoupling proteins [26]. Cold temperature can activate BAT in adult humans, irrespective of age and gender [27]. Over thirty years ago, research suggested that obesity could be treated by exercise in the cold [28]. On the other hand, in a hot environment, the propensity for feeding is diminished [5], and air conditioning may contribute to rising obesity because the body expends less energy in temperature ranges associated with climate-controlled settings (via postural adjustments and evaporative cooling) [1, 29]. Furthermore, the rising trend in central air conditioning could provide an incentive for people to remain indoors and exercise less [7]. We found a non-linear relationship between mean house temperature and BMI and fasting glucose levels at follow-up; a 2-fold increased risk for both incident obesity and hyperglycemia was estimated in subjects living at an indoor temperature greater than 20°C. It might be hypothesized that metabolic processes are favorably affected by an ambient temperature within the thermal neutral zone, i.e., not requiring energy expenditure to be allocated to maintaining a constant body temperature [1]. However, no evidence exists to support this and socio-economic factors might confound these associations.

In our cohort, air conditioning use, although associated with more than three-fold higher incidence of both obesity and hyperglycemia, showed a lower impact on these conditions compared to other risk factors.

Diet and exercise

Several dietary factors that increase the risk for obesity and diabetes have been identified; among them, a reduced fiber intake has shown to play a strong predictive role for the incidence of type 2 diabetes [30], in line with our results. Meal consumption in restaurants was unusual in this middle-aged cohort, as less than 10% of the study subjects regularly consumed food away from home. Those who did consume food away from home did so with the following distribution: 47% pizzerias, 35% full-service restaurants and 18% fast-food restaurants. Compared to food prepared at home, restaurant food has high energy density, more fat and high glycemic load, and portion sizes are usually larger [6]. It has been shown that a higher ratio of fast-food to full-service restaurant density was associated with higher BMI and risk of obesity [31]. Furthermore, people find it difficult to estimate the caloric contents of food items at restaurants and tend to underestimate their energy content [32]. Due to the low number of subjects regularly consuming meals in restaurants, our study did not have sufficient statistical power to detect risk differences by restaurant type. Nevertheless, the associations between the incremental number of meals consumed away from home and incident obesity and hyperglycemia were strong and largely independent of other potentially confounding lifestyle factors. The incremental rises in BMI and glucose levels at follow-up were greatest in patients eating ≥4 restaurant meals/week and the values were comparable to the data obtained for fast-food consumption [33]. These results are of potential interest for public programs aimed at reducing the diabetes and obesity epidemics.

Physical activity plays a central role in diabetes and obesity prevention [34], and reduced exercise at baseline predicted the incidence of obesity in our cohort. Television watching was positively associated with both incident obesity and hyperglycemia, but this relationship was not significant in the multivariate model, probably due to the predominant roles of reduced exercise and un-healthy dietary factors in obesity and hyperglycemia respectively; both of these conditions are strongly associated with increased amounts of time watching television [35].

Other possible contributors

Even if incident obesity and hyperglycemia were 5- and 2-fold higher, respectively, in users of antidepressant/antipsychotic drugs, the associations were smaller and no longer statistically significant in the multivariate model, suggesting that the crude association was confounded by other risk factors. However, due to the low prevalence of antidepressant/antipsychotic drug users in our cohort (95/1597; 5.9%), the study lacked sufficient statistical power to detect small risks.

Limitations and strengths

A potential limitation of this study was the reliance on self-reported diet and other lifestyle factors. In addition, because this was an observational study, the possibility of confounding by unmeasured variables cannot be excluded. Socioeconomic status, which was not analyzed, might be a potential confounder. Nevertheless, we introduced education level into the multivariate models; this variable is a reliable indicator of socioeconomic status because it is stable, established in early adulthood and not modified by chronic disease [36]. Random misclassification and measurement errors in our prospective study would result in attenuated estimates of the strengths of the association with the outcome variables.

Finally, our results were limited to middle aged individuals, many of whom lived in rural areas and had low levels of education. However, the population-based cohort and the biological plausibility of our results, which are in accordance with previous studies each analyzing a single contributor, lend support to our conclusions.

The strengths of this study were the fact that a large proportion of subjects were enrolled from a defined community and its focus on multiple novel explanations for incident obesity and hyperglycemia at once.

Conclusions

Sleep restriction, higher home temperature and regular consumption of restaurant meals might represent lifestyle contributors to the obesity and hyperglycemia epidemics. Avoiding these behaviors could be a potential adjunctive nonpharmacologic strategy for preventing the obesity and hyperglycemia epidemics.

Acknowledgments: This study was supported by a grant from: Regione Piemonte, 2008.

Conflicts of interest: none

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Figure 1 Baseline environmental characteristics and BMI at follow-up, by group of increment of the variables

Legend to Figure 1:

Upper -lower values

75th-25th percentile

median

Restaurant foods: group 1: no consumption of restaurant foods, *n*=1165; group 2: 1-3/week, *n*=78; group 3: ≥4/week, *n*=39

House temperature: first tertile ≤18°, *n*=354; second tertile=>18°<20°, *n*=401; third tertile ≥20°, *n*=527

Television hours: group 1: 0-1 h/day, *n*=533; group 2: 2-3 h/day, *n*=546; group 3: >3 h/day, *n*=203

Sleep hours: group 1: ≤6.5 h/day, *n*=309; group 2: =7h/day, *n*=456; group 3: >7h/day, *n*=517

METS: group 1: <18h/week, *n*=410; group 2: ≥18h/week <24h/week, *n*=411; group 3:≥24h/week, *n*=461

Fiber intake (g/day): group 1: first tertile <16.5g/day, *n*=447; group 2: second tertile ≥16.5g/day <23.5g/day, *n*=413; group 3: third tertile ≥23.5g/day, *n*=422

Saturated fat intake (% energy): group 1: first tertile <10.5%, *n*=428; group 2: second tertile ≥10.5%<12.7%, *n*=426; group 3: third tertile ≥12.7%, *n*=428

Figure 2. Baseline environmental characteristics and fasting glucose values at follow-up, by group of increment of the variables

Legend to Figure 2:

Upper -lower values

75th-25th percentile

median

Restaurant foods: group 1: no consumption of restaurant foods, *n*=893; group 2: 1-3/week, *n*=55; group 3: ≥4/week, *n*=31

House temperature: first tertile ≤18°, *n*=250; second tertile=>18°<20°, *n*=325; third tertile ≥20°, *n*=404

Television hours: group 1: 0-1 h/day, *n*=413; group 2: 2-3 h/day, *n*=427; group 3: >3 h/day, *n*=139

Sleep hours: group 1: ≤6.5 h/day, *n*=252; group 2: =7h/day, *n*=320; group 3: >7h/day, *n*=407

METS: group 1: <18h/week, *n*=312; group 2: ≥18h/week <24h/week, *n*=318; group 3:≥24h/week, *n*=349

Fiber intake (g/day): group 1: first tertile <16.5g/day, *n*=334; group 2: second tertile ≥16.5g/day <23.5g/day, *n*=335; group 3: third tertile ≥23.5g/day, *n*=310

Saturated fat intake (% energy): group 1: first tertile <10.5%, *n*=316; group 2: second tertile ≥10.5%<12.7%, *n*=321; group 3: third tertile ≥12.7%, *n*=342

Table 1. Baseline characteristics according to obesity development at follow-up

	Obesity yes	Obesity no	P
Number	103	1179	
Age (years)	54.8±6.1	54.3±5.6	0.37
Males (%)	52.4	46.1	0.22[1]
BMI (kg/m^2)	28.4±2.0	24.5±2.7	<0.001
Waist circumference (cm)	95.0±8.9	86.9±10.5	<0.001
Fasting glucose (mmol/L)	5.8±1.3	5.7±1.6	0.35
Energy intake (kcal/day)	2173.9±773.7	2072.8±656.7	0.14
Fat (% energy)	35.6±6.0	34.9±5.9	0.23
Saturated fat (% energy)	13.6±5.0	11.8±2.9	<0.001
Carbohydrates (%energy)	47.7±7.3	48.7±7.1	0.16
Fiber (g/day)	18.1±7.9	21.4±9.6	0.005
Actual smoking (%)	23.3	24.6	0.77[1]
Alcohol (g/day)	17.9±28.0	17.3±26.4	0.72[2]
Number of restaurant food intake/week	1.2±1.8	0.2±0.9	<0.001[2]
METS (h/week)	17.3±7.0	22.1±9.4	<0.001
Television watching (h/day)	2.8±2.0	1.9±1.6	<0.001[2]
Use of antidepressant/ antipsychotic drugs (%)	12.6	2.5	<0.001[1]
Air conditioning use (%)	16.5	5.0	<0.001[1]
Hours of sleep/day	6.3±1.4	7.2±1.1	<0.001
Mean house temperature (°C)	20.8±2.1	19.4±1.6	<0.001

Education level (%):			
Primary school	78.6	72.3	
Secondary school	13.6	19.1	
University	7.8	8.6	0.34[1]
Living in a rural area (%)	35.9	39.8	0.44[1]

P-values calculated by *t*-Student test

[1] p-values calculated by Chi-square test

[2]p-values calculated by Mann-Whitney test

Table 2. Baseline characteristics according to hyperglycemia development at follow-up

	Hyperglycemia yes	Hyperglycemia no	P
Number	174	805	
Age (years)	53.6±5.3	53.9±5.7	0.53
Males (%)	47.1	36.5	0.009[1]
BMI (kg/m^2)	26.2±4.6	25.4±4.2	0.02
Waist circumference (cm)	90.8±13.0	87.2±12.1	<0.001
Fasting glucose (mmol/L)	5.3±0.3	5.0±0.4	<0.001
Energy intake (kcal/day)	2130.9±706.5	2055.9±655.0	0.18
Fat (% energy)	34.5±5.6	35.4±6.0	0.06
Saturated fat (% energy)	12.6±4.3	11.9±2.7	0.008
Carbohydrates (%energy)	49.6±6.7	48.2±7.2	0.02
Fiber (g/day)	18.5±9.2	21.7±9.2	<0.001
Actual smoking (%)	19.0	24.3	0.13[1]
Alcohol (g/day)	18.5±24.4	13.4±24.3	<0.001[2]
Number of restaurant food intake/week	0.9±1.7	0.1±0.7	<0.001[2]
METS (h/week)	19.8±8.7	21.9±9.4	0.008
Television watching (h/day)	2.5±1.8	1.8±1.6	<0.001[2]
Use of antidepressant/ antipsychotic drugs (%)	6.3	3.2	0.05[1]
Air conditioning use (%)	10.9	4.6	0.001[1]
Hours of sleep/day	7.0±1.2	7.2±1.2	0.07
Mean house temperature (°C)	20.6±2.4	19.4±1.3	<0.001

Education level (%):			
Primary school	75.3	72.5	
Secondary school	17.8	18.4	
University	6.9	9.1	0.62[1]
Living in a rural area (%)	44.8	38.8	0.14[1]

P-values calculated by *t*-Student test

[1] p-values calculated by Chi-square test

[2] p-values calculated by Mann-Whitney test

Table 3. Association between baseline variables and obesity at follow-up in a logistic regression model: crude (left) and adjusted (right)

	OR	95%CI	P	OR[1]	95%CI	P
Male sex	1.29	0.86-1.93	0.22	1.02	0.57-1.82	0.95
BMI (kg/m^2)	2.75	2.51-3.03	<0.001	2.50	2.03-3.05	<0.001
Saturated fat (% energy)	1.14	1.08-1.20	<0.001	1.07	0.99-1.15	0.09
Fiber (g/day)	0.96	0.93-0.98	<0.001	0.99	0.96-1.02	0.60
METS (h/week)	0.93	0.91-0.96	<0.001	0.94	0.91-0.98	<0.001
Number of restaurant food intake/week	1.65	1.45-1.86	<0.001	1.47	1.21-1.79	<0.001
Television watching (h/day)	1.32	1.18-1.46	<0.001	1.07	0.91-1.27	0.42
Antidepressant/antipsychotic drug use (%)	5.61	2.82-11.2	<0.001	1.56	0.52-4.63	0.43
Air conditioning use (%)	3.81	2.12-6.85	<0.001	2.21	0.93-5.27	0.07
Hours of sleep/day	0.53	0.45-0.63	<0.001	0.70	0.57-0.86	<0.001
First tertile house temperature (°C)[2]	1			1		
Second tertile house temperature (°C)	0.65	0.31-1.35	0.24	1.05	0.44-2.48	0.92
Third tertile house temperature (°C)	3.14	1.82-5.43	<0.001	2.06	1.02-4.16	0.04
Primary school (%)	1.50	0.91-2.46	0.11	1.41	0.73-2.72	0.30

[1] Odd ratios adjusted for all the variables listed in the table

[2] Tertiles of house temperature were: first tertile ≤18°, *n*=354; second tertile=>18°<20°, *n*=401; third tertile ≥20°, *n*=527

Table 4. Association between baseline variables and hyperglycemia at follow-up in a logistic regression model: crude (left) and adjusted (right)

	OR	95%CI	P	OR[1]	95%CI	P
Male sex	1.55	1.11-2.16	0.009	1.12	0.73-1.70	0.61
Glucose (mmol/L)	23.7	11.6-48.4	<0.001	20.3	9.37-44.1	<0.001
Saturated fat (% energy)	1.07	1.02-1.12	0.009	1.00	0.93-1.07	0.99
Fiber (g/day)	0.96	0.94-0.98	<0.001	0.97	0.95-0.99	0.01
METS (h/week)	0.98	0.96-0.99	0.008	0.99	0.97-1.01	0.21
Number of restaurant food intake/week	1.66	1.44-1.91	<0.001	1.49	1.26-1.75	<0.001
Television watching (h/day)	1.27	1.16-1.39	<0.001	1.10	0.98-1.23	0.11
Antidepressant/ antipsychotic drug use (%)	2.02	0.98-4.18	0.06	0.93	0.36-2.34	0.87
Air conditioning use (%)	2.54	1.42-4.55	0.002	1.47	0.73-2.96	0.28
Hours of sleep/day	0.88	0.77-1.01	0.07	1.06	0.91-1.24	0.43
First tertile house temperature (°C)[2]	1			1		
Second tertile house temperature (°C)	0.72	0.42-1.22	0.22	0.76	0.43-1.35	0.35
Third tertile house temperature (°C)	2.82	1.81-4.37	<0.001	1.95	1.17-3.26	0.01
Primary school (%)	1.15	0.79-1.68	0.46	1.07	0.69-1.66	0.76

[1] Odd ratios adjusted for all the variables listed in the table, plus BMI and alcohol intake at baseline

[2] Tertiles of house temperature were: first tertile ≤18°, *n*=250; second tertile=>18°<20°, *n*=325; third tertile ≥20°, *n*=404

Figure 1. Baseline environmental characteristics and BMI at follow-up, by group of increment of the variables

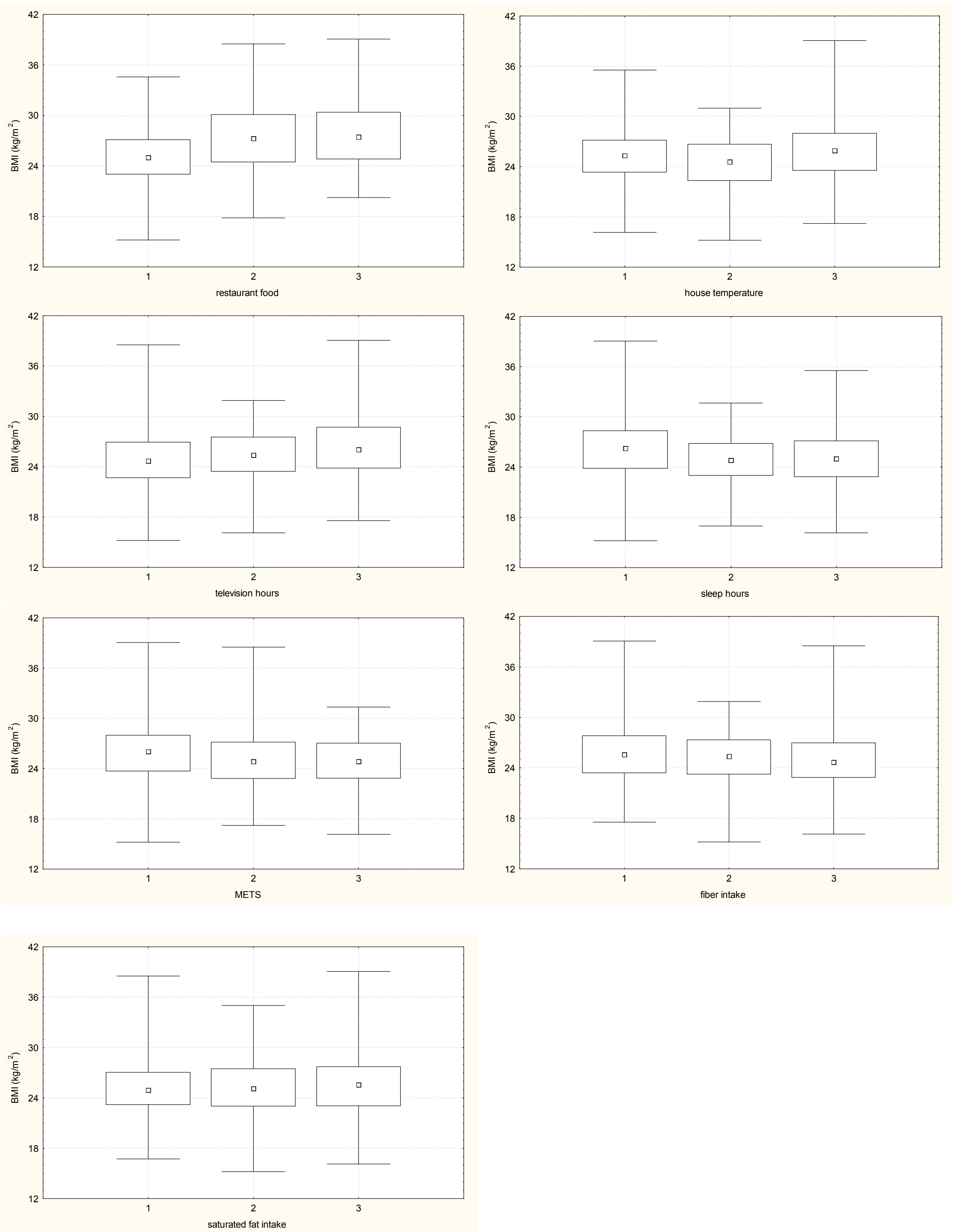

Figure 2. Baseline environmental characteristics and fasting glucose at follow-up, by group of increment of the variables